# **Electrocardiogram Basics for the Busy Pediatrician**

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#### Introduction

n electrocardiogram (ECG) is the representation of the electrical activity of the heart recorded from the surface of the body by the use of electrodes linked to a galvanometer. The 12-lead ECG is an invaluable tool for assessing the heart. Clinical indications for performing it include any symptoms referable to the cardiovascular system including palpitations, syncope, chest pain, cyanosis, or an abnormal cardiovascular examination. An ECG might also be helpful in screening for genetic cardiac disorders, for evaluating electrolyte disturbances, and intoxications. An ECG is irreplaceable for rate and rhythm concerns and remains essential in the follow-up evaluation of patients with structural heart disease.

#### **Technique**

The 12-lead ECG includes the recording from the standard bipolar leads I, II, and III; the augmented leads aVF, aVL, and aVR; and the standard unipolar precordial leads V1 to V6. The rightsided leads V3R, V4R, and V7 may be recorded resulting in a 15-lead ECG. In addition, a longer recording of a single lead (usually lead II) is obtained to analyze the rhythm more precisely. In a patient known to have dextrocardia, the standard precordial leads should be positioned over the right chest in a mirror image manner. The limb leads may be most stably placed on the flat area of the shoulders and the legs during recording. Neonatal electrodes may be obtained by trimming the adult-sized electrodes. Cleaning the neonatal skin with alcohol will lower the high skin impedance associated with vernix.

Clin Pediatr. 2006;45:597-604

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DOI: 10.1177/0009922806291004 © 2006 Sage Publications

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#### Pediatric 12-Lead ECG Analyses

Standardization

In the standard recording at a speed of 25 mm/sec, each small box along the X-axis represents 0.04 seconds. Each large square is 0.2 seconds (Figure 1). Each small square along the Y-axis represents 0.1 millivolt at standard amplification. Half standardization is used to attenuate complexes that would otherwise overlap each other (as in QRS complexes in ventricular hypertrophy). A fully standardized ECG should also be recorded to analyze significant ST-T changes that might be masked on the half standard ECG. Lead II is chosen for most ECG "interval" measurements, as it is parallel to the long axis of the heart.

#### Heart Rate

To determine heart rate from the ECG, use the onset of the QRS complex rather than the peak of the R wave for a more accurate measurement. The following several methods may be used:

*Method 1*: Count the number of small squares between the R-R intervals and divide 1500 by the number of small squares.

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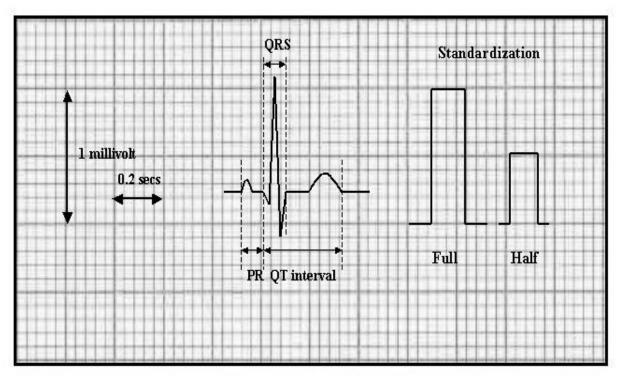


Figure 1. ECG standardizations and intervals.

Method 2: Count the number of large squares between the R-R intervals and divide 300 by the number of large squares (Figure 2).

Method 3: Count and multiply the number of QRS complexes on the 12-lead ECG paper by 6 to obtain the heart rate (as the length of ECG paper is 250 mms and the paper speed is 25 mm/sec, it takes 10 seconds to record an ECG).

#### Rhythm

Normal sinus rhythm is defined by a P wave preceding each QRS at an appropriate interval. The P wave axis should be between 0° and 90° (determined as it is for the QRS complexes). Sinus arrhythmia is defined as a normal variation in the heart rate associated with respiration while

maintaining the characteristics of normal sinus rhythm (Figure 3). Sinus bradycardia is physiologic in conditioned athletes. Heart block occurs because of a delay in conduction of impulse at various locations in the electrical pathway of the heart. Refer to Table 1 for types and causes of heart block.

#### Electrical Axis

Axis of ventricular depolarization is determined by the QRS complex axis. The hexa-axial reference system (Figure 4) helps in determining the frontal axis of the electrical activity of the heart. Lead I and aVF may be used to determine a general QRS axis. The net QRS deflection in lead I and aVF are plotted on the hexa-axial system. Normal QRS axis lies between 0° and 110°, although nor-

mal newborns may have a right axis deviation (90° to 180°). The northwest (NW) axis or indeterminate axis is defined for QRS axis between –100° and +210°. It could represent either extreme right or left axis deviation. In the presence of a mean vector in the NW axis, extreme left axis deviation exists when there is an initial q wave in lead I and extreme right axis deviation exists when there is an initial q wave in lead aVF (Table 2).

#### P Wave

Represents atrial depolarization. A P wave originating in the sinus node (normal) is upright in leads I and aVF. A low right atrial rhythm may sometimes be seen and is a normal variant. Atrial activation in this instance begins

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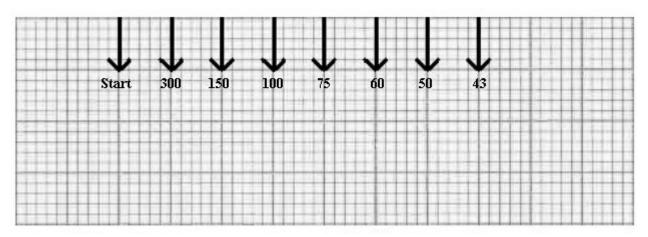


Figure 2. Determination of heart rate.

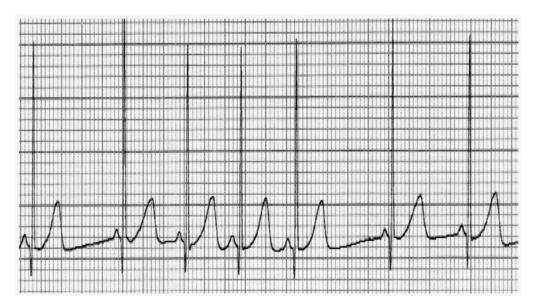


Figure 3. Sinus arrhythmia.

from the low right atrium and proceeds to depolarize the remaining atrial mass with a mean vector traveling to the left and upward. The resultant P wave is negative in aVF and positive or isoelectric in lead I.

A P wave taller than 2.5 small squares in lead II denotes right atrial enlargement in all ages (Figure 5) and a P wave wider than 2 small squares in lead II

represents left atrial enlargement (Figure 6). A P wave taller and wider than 2.5 and 2 small squares, respectively, denotes biatrial enlargement. A biphasic P wave is normal in lead V1, but the terminal negative component should be less than 1 small square in duration and less than 1 small square in depth. If not, the findings might represent left atrial enlargement.

#### QRS Complex

Represents ventricular depolarization.

#### **ORS** duration

This is preferably measured in a limb lead that has a Q wave. A QRS complex measuring more than 0.07 sec in less than 3 years of age, more than 0.08 sec in 3 to 12 years of age, and more than 0.1 sec in children older than 12 years

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Table 1			
HEART BLOCKS: TYPES AND CAUSES			
Type of Heart Block	ECG Findings	Causes	
First degree heart block	Prolonged PR interval	Normal variant, increased vagal tone, acute rheumatic fever, myocarditis/cardiomyopathy, drugs causing conduction delay (digoxin, beta-blockers, etc.), cardiac surgery	
Second degree heart block	Type I: Progressive prolongation of PR interval until a nonconducted P wave occurs	Same as first degree heart block	
	Type II: Constant PR interval with intermittent nonconducted P wave	Very rare in pediatric patients.  More serious than type I and can progress to complete heart block	
Third degree heart block	Complete heart block with atrial (P wave) and ventricular (QRS complex) activities independent of each other	Congenital complete heart block, cardiac surgery, myocarditis, myocardial infarction, drugs	

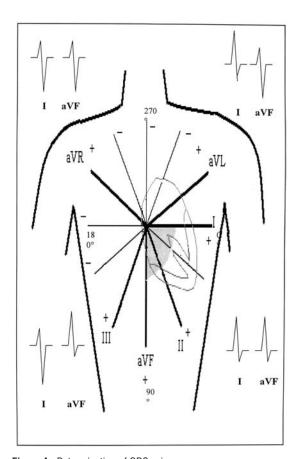
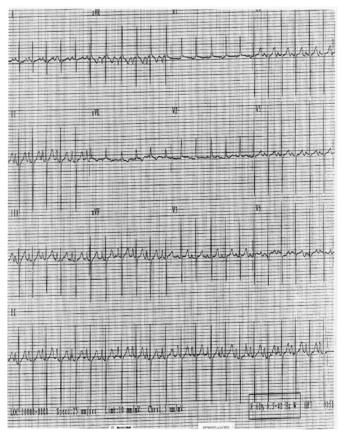


Figure 4. Determination of QRS axis.



**Figure 5.** Right atrial enlargement (tall peaked P wave) with right ventricular hypertrophy (pure R wave in V1 and deep S wave in V6).

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#### Table 2

#### CAUSES OF QRS AXIS DEVIATION

Axis Deviation	Causes
1. Right axis deviation	Normal in neonates RVH RBBB Certain forms of congenital cardiac defects
2. Left axis deviation	Congenital cardiac defects AV canal defects Tricuspid atresia LBBB Normal in approximately 1% of the population

RVH, right ventricular hypertrophy; RBBB, right bundle branch block; LBBB, left bundle branch block.

#### Table 3

## CAUSES OF RIGHT BUNDLE BRANCH BLOCK (RBBB) AND LEFT BUNDLE BRANCH BLOCK

Causes of RBBB Cardiac surgery especially VSD closure, TOF repair

ASD (incomplete RBBB, r < R')
Patients with ventricular pacemaker
Occasionally in normal children

Causes of LBBB Cardiac surgery especially involving LV outflow tract

Hypertrophic cardiomyopathy

Myocarditis

VSD, ventricular septal defect; TOF, Tetrology of Fallot; ASD, atrial septal defect; LV, left ventricular; RBBB, right bundle branch block: LBBB, left bundle branch block.

of age is considered wide complex. The causes of a wide QRS complex include bundle branch block, complexes originating in the ventricles, paced rhythms, ventricular preexcitation, drug effects, and electrolyte abnormalities. A wide complex QRS with rSR' pattern in lead V1 is suggestive of a right bundle branch block. An RSr' pattern with normal QRS duration can be seen in about 5% to 7% of normal chil-

dren. A left bundle branch block pattern is rarely seen in children (Table 3). The ECG is unreliable for assessing ventricular hypertrophy and ischemia in the presence of a bundle branch block.

#### QRS morphology

The QRS morphology should be systematically analyzed in the limb leads followed by the precordial leads. A q wave is a negative deflection before any positive deflection. The first positive deflection is defined as R wave and subsequent positive deflections are defined as R', R", and so on. The negative deflection after the first positive deflection is defined as the S wave.

A q wave in the left precordial leads represents the initial depolarization of the interventricular septum. A normal q wave is less than 1 small square in duration and less than 25% of the QRS amplitude in depth. The q waves are normally seen in leads II, III, and aVF and in left precordial leads. Pathologic q waves are more than 5 mm in depth and more than 1 small square in duration. Pathologic q waves in lead I, aVL, and the left precordial leads are suggestive of an anterolateral wall infarction. It is diagnostic of anomalous origin of the left coronary artery from the pulmonary artery in children.

Morphological changes of ventricular hypertrophy are described in Table 4 and Figures 5 and 7. None of these findings are diagnostic of ventricular hypertrophy, but their presence would warrant clinical correlation. Low QRS voltage is said to be present when the QRS amplitude is less than 5 mm in all limb leads and less than 10 in all precordial leads. It may be seen with myocarditis, pericardial effusion, generalized anasarca, chronic obstructive pulmonary disease, or in patients with anorexia nervosa.

#### T Wave

T waves represent ventricular repolarization. T waves are normally inverted in the pediatric age group in the precordial leads V1 to V3 and are always upright in lead V6. In the first 7 days of life, the T wave in right-sided precordial leads undergoes multiple polarity changes. This should be

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#### Table 4

#### **ECG CRITERIA FOR VENTRICULAR HYPERTROPHY**

#### Criteria for LVH

- R wave amplitude in lead V6 plus S wave amplitude in lead V1 greater than the upper limits of normal for age, or greater than 25 mm each
- T wave inversion in the inferior (II, III, and aVF) and lateral (V5 and V6) leads (strain pattern)
- · Left axis deviation
- · Abnormally prominent q waves in lateral leads

#### Criteria for RVH

- R wave in lead V1 and S wave in lead V6 greater than the upper limits of normal for age
- Upright T waves in lead V1 after 7 days of age (strain pattern)
- qR pattern in lead V1

LVH and RVH, left and right ventricular hypertrophy.

kept in mind while analyzing neonatal ECGs. In adolescence, T wave polarity progressively changes, becoming upright in the right precordial leads and remaining upright in the left precordial leads. T waves taller than 7 mm in a limb lead and more than 10 mm in a precordial are considered tall. Tall peaked T waves suggest left ventricular volume overload or hyperkalemia.

#### PR Interval

The PR interval represents conduction through the atria and the AV node-His Purkinje system. The normal duration varies with age and is 0.1 to 0.2 seconds. PR interval is short in low atrial rhythm and with preexcitation. Preexcitation is defined as the premature activation of part of the ventricle due to transmission of impulses along an accessory pathway that is not subject to the normal delay at the AV node. PR interval is short and associated with a delta wave (representing preexcitation) in patients with Wolff-Parkinson-White syndrome (Figure 8).

#### QT Interval

QT interval represents ventricular depolarization and subsequent repolarization. It is measured from the beginning of the QRS complex to the end of T wave. Normal QT interval varies

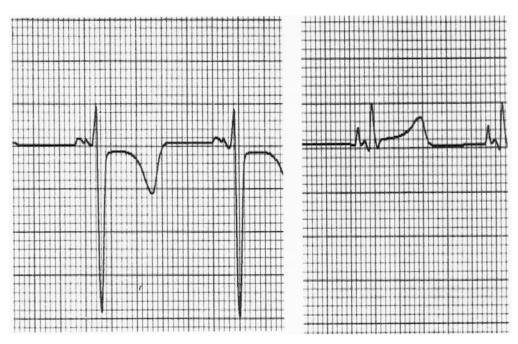


Figure 6. Left atrial enlargement (wide m-shaped P wave).

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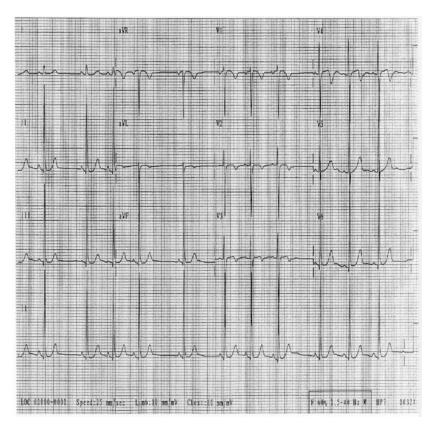


Figure 7. Left ventricular hypertrophy (tall R wave in V6 > 25 mm).

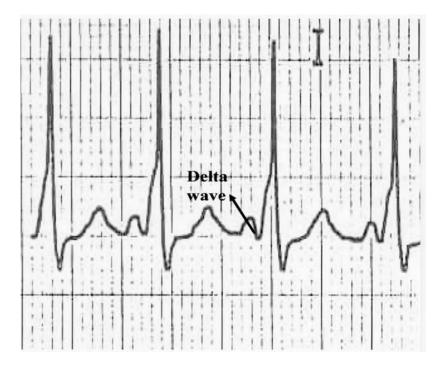


Figure 8. Delta wave in WPW syndrome.

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with heart rate and, therefore, should be corrected for heart rate (QTc). The most commonly used formula for rate correction is:

$$QTc (sec) = \frac{Measured QT (sec)}{-R-R interval (sec)}$$

Normal QTc is less than 450 msec. Prolonged QTc is seen in patients with long QT syndrome, electrolyte imbalance (especially hypocalcemia or hypomagnesemia), drug effect (especially tricyclic antidepressants, cisapride, macrolide antibiotics, procainamide, quinidine, and so on), and organophosphate poisoning. Assessment of QTc prolongation in patients with bundle branch block pattern is unreliable. Prolonged QTc is a risk factor for ventricular arrhythmias as the vulnerable period of the ventricular muscle is prolonged. In patients with "long QT syndrome," the morphology of the QT segment and or the T waves is usually abnormal.

#### ST Segment

The ST segment is from the end of ventricular depolarization (QRS complex) to the beginning of ventricular repolarization (T wave). It is normally isoelectric. A deviation by more than 2 small squares in the precordial leads is pathologic. A common normal finding in black adolescent boys is ST segment elevation seen in mid and left precordial leads. This is suggestive of early repolarization and is usually considered a benign condition. Generalized ST segment elevation with concavity upward is suggestive of acute pericarditis. The ST segment elevation can progress to generalized T wave inversion with evolving stages of pericarditis.

#### Premature Complexes

Premature atrial complex (PAC)

This is also known as premature atrial beat and does not originate from the sinus node. The P wave usually has a different contour and axis. PACs can result in normally conducted QRS complex, wide QRS complex (aberrant conduction), or can be blocked at the AV node, depending on the degree of prematurity. PACs can present as an irregular rhythm. PACs are usually benign and do not require any treatment.

## Premature junctional complex (PJC)

A PJC is a premature beat originating in the AV junction, not preceded by a P wave. The QRS morphology in a PJC resembles a sinus beat. PJCs are usually benign.

## Premature ventricular complex (PVC)

A PVC is a premature beat originating from the ventricle not preceded by a P wave. The PVC has a different morphology and is wider compared to a normal QRS complex. PVCs can also present as an irregular rhythm. Isolated PVCs are usually benign. A finding of frequent PVCs, PVCs with varying morphology, or more than 3 PVCs in a row warrants further investigation.

#### **Red Flags**

#### Chest Pain

In a patient with suspected cardiac origin of chest pain, look for pathologic q waves, ST-T abnormalities, arrhythmias, or hypertrophy.

#### **Palpitations**

In a patient with palpitations, arrhythmias, conduction distur-

bances/blocks, QTc abnormalities, and evidence of preexcitation should be considered.

#### Syncope

In a patient with syncope, evaluate QTc interval, T wave morphology (such as T wave alternans—alternating morphology or axis of the T wave), biphasic T waves, or notched T waves as indicators of the presence of long QT syndrome. Also consider arrhythmias, conduction disturbances/blocks, and evidence of hypertrophy.

#### Ingestions

In patients with ingestion of medications or drugs with cardiotropic effects look for changes in cardiac intervals, arrhythmias, and conduction disturbances.

#### Conclusion

Most pediatricians express anxiety and discomfort when required to interpret an ECG because of unfamiliarity with the transitional changes in pediatric ECG and concern about missing important information. A systematic approach toward routinely reading and interpreting ECGs, as outlined above, will help gain confidence in reading and interpreting ECGs. If there is any suspicion of an unusual finding or an abnormality in an ECG, it is imperative to obtain an opinion of a pediatric cardiologist within a reasonable period of time.

#### Suggested Reading

Garson A, Bricker TJ, Fischer DJ, et al. *The Science and Practice of Pediatric Cardiology*. 2nd ed. Lippincott, Williams and Wilkins, Dallas, TX; 1998.

Park MK. Pediatric Cardiology for Practitioners. 4th ed. Mosby, Inc, San Antonio, TX; 2002.